

Risks of Consumption of Contaminated Seafood: The Quincy Bay Case Study

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A recent EPA-sponsored study of sediment and seafood contamination in Quincy Bay revealed elevated levels of several complex organic pollutants frequently of concern in human health assessments. A seafood consumption risk assessment was conducted using data from samples collected in Quincy Bay in the methodology developed for EPA's Office of Marine and Estuarine Protection for such assessments. Results showed estimated plausible, upperbound excess cancer risks in the 10^{-5} to 10^{-2} range. These results are comparable to those found in other seafood contamination risk assessments for areas where consumption advisories and fishing restrictions were implemented. Regulatory response included consumption advisories for lobster tomalley (hepatopancreas) and other types of locally caught seafood. Uncertainties inherent in seafood risk assessment in general and for the Quincy Bay case are discussed, along with implications for further action.

Background

Quincy Bay, located on the Massachusetts coast just south of Boston, is known as the "Winter Flounder Capital of the World." In addition to being a popular recreational fishing center, Quincy Bay is also the receiving water for discharges from the 130+ mgd (million gallons per day) Nut Island wastewater treatment plant and from a major combined sewer overflow (CSO) on Moon Island. As part of the Massachusetts Water Resources Authority system, both of these facilities handle large quantities of wastewater from the greater Boston area.

Concern about environmental degradation in Quincy Bay resulting from these point source discharges led U.S. Congressman Brian Donnelly, whose District includes the City of Quincy, to get legislation passed directing the U.S. Environmental Protection Agency (EPA) to undertake a study to determine the types and concentrations of pollutants and the extent of sludge in Quincy Bay. The study was also mandated to include an evaluation of the public health risks associated with Quincy Bay sediments.

To accomplish the study objectives, the Quincy Bay Study was divided into five tasks as follows: task I, review of historical data for characterization of Quincy Bay contamination; task II, sampling and analysis for evaluation of sediment contamination; task III, sampling and analysis for evaluation of fish and shellfish contamination and histopathology; task IV, analysis of fish and shellfish consumption and assessment of risk to public health; and

task V: synthesis of findings into a summary report containing EPA recommendations. EPA Region I managed the study and provided technical oversight, Metcalf & Eddy was contracted to complete tasks I, IV, and V, and the EPA Environmental Research Laboratory at Narragansett, Rhode Island, performed tasks II and III.

The Quincy Bay Study commenced in the Spring of 1987, with the majority of the sampling taking place during May 1987. Sediment cores and surface scoop samples were taken for chemical analysis. Winter flounder, lobster, and softshell clams were collected for chemical and histopathological analysis. Data and draft reports underwent extensive peer and agency reviews before final public release of the findings in June of 1988. Implementation of the study recommendations is a continuing process.

Task I provided a detailed assessment of historical data for Quincy Bay and Boston Harbor. Results of task II were that Quincy Bay sediments are contaminated, similar in levels to Boston Harbor. Organic chemicals were generally high, while metals were elevated but low relative to the rest of Boston Harbor. The highest levels were found in five sediment depositional areas.

Analysis of tissue residues in task III revealed contamination with organics, especially polychlorinated biphenyls (PCBs). Lobster tomalley (hepatopancreas) had extremely high levels of PCBs, averaging about 30 ppm. Histopathological investigations found 83% of winter flounder had either gross or microscopic evidence of liver disease; 23% had liver neoplasms. In addition, clams had pathology in 80% and viruses in 51% of individuals examined. Six percent of oysters deployed for 40 days developed abnormalities.

The task IV health risk assessment was performed using tissue residue data from task III. The risk assessment was the most controversial part of the study, and the rest of this paper examines the risk assessment methodology and results and the risk management decisions and policy implications.

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Table 1. Summarized contaminant

Chemical identified	IRIS	HEA	FDA limits, ppm ^b	Carcinogenic potency factor, mg/kg/day		EPA weight of evidence ^c	Reference dose, mg/kg/day ^d	Reference	Toxicity rating ^e
				Oral	Inhaled				
Elements/metals									
Cadmium	Y	Y	—	—	6.10E+00	B1	2.90E-04	(13)	10
Chromium	Y	Y	—	—	4.10E+01	A	5.00E-03	(13)	8
Copper	N	Y	—	—	—	D	3.70E-02	(13)	5
Mercury ^f	N	Y	1.0	—	—	D	2.0E-03	(14)	7
Lead ^f	N	Y	—	—	—	C	1.40E-03	(14)	10
Organic compounds									
Chlordane (total) ^g	Y	Y	0.3	1.30E+00	—	B2	5.00E-05	(14)	2
α -Chlordane	Y	Y	0.3	1.30E+00	—	B2	5.00E-05	(14)	2
γ -Chlordane	Y	Y	0.3	1.30E+00	—	B2	5.00E-05	(14)	2
<i>pp</i> -DDD	N	N	5.0	3.40E-01	—	—	5.00E-04	(13)	2
<i>pp</i> -DDE	N	N	5.0	3.40E-01	—	B2	5.00E-04	(13)	2
<i>pp</i> -DDT	N	N	5.0	3.40E-01	—	B2	5.00E-04	(13)	2
Hexachlorobenzene	N	Y	—	1.69E+00	—	B2	8.00E-04	(13)	3
Hexachlorocyclohexane	Y	Y	—	—	—	B2	—	—	—
α -HCH	—	—	—	6.30E+00	—	B2	3.00E-04	(13)	2
γ -HCH (indane)	Y	N	—	1.33E+00	—	B2/C	3.00E-04	(13)	2
PAHs (total)	Y	Y	—	1.15E+01	6.11E+00	—	—	—	—
PCBs (total)	N	N	2.0	2.60E+00	—	B2	1.00E-04	(15)	—
Aroclor 1242	N	Y	—	—	—	—	—	—	—
Aroclor 1254	N	N	—	—	—	—	—	—	—

^aData are presented as microgram/gram wet weight, converted from Gardner and Pruell (1). Means were calculated using detection limits for undetected detected.

^bFrom Tetra Tech, Inc. (12).

^cEPA weight of evidence is the rating that qualifies the level of evidence that supports designating a chemical a human carcinogen. Weight of evidence

^dReference dose or acceptable intake-chronic level is the long-term acceptable intake level for noncarcinogenic effects. Values were obtained from the Superfund

^eToxicity ratings are unitless integers ranging from 1 to 10 and corresponding to various severity levels of effects.

^fData correspond to inorganic compound values.

^gSame carcinogenic potency factor value used for both chlordane isomers.

Risk Assessment Methodology

Contaminant levels detected in seafood from Quincy Bay (1) were evaluated to investigate potential carcinogenic and noncarcinogenic public health implications from ingestion of the seafood. The methodology used was provided in the *Guidance Manual for Assessing Human Health Risks from Chemically Contaminated Fish and Seafood* (2), which is consistent with the other widely applied EPA risk assessment guidelines. Specifically, the methodology for carcinogen assessment is based on the linearized, multistage modeling concept for carcinogenesis. For assessment of noncarcinogenic effects, the methodology uses a hazard index approach based on the ratio of the calculated study-specific ingested contaminant dose to EPA's standard reference doses (RfDs) for the same contaminants. Only the carcinogen assessment results are discussed here.

The contaminants included in the study were organic compounds and metals measured by EPA in sediment and seafood samples from Quincy Bay in 1987 (1). For hazard identification (Table 1), the Integrated Risk Information System (IRIS) was used extensively to obtain carcinogenic potency factors (CPF). The standard CPFs used were derived by EPA using a linearized, multistage model and reflect a plausible upper-bound value. They are used to convert calculated dose to plausible upper-bound incremental cancer risk.

Three species were chosen for chemical contaminant evaluation from Quincy Bay: winter flounder (*Pseudopleuronectes americanus*), softshell clams (*Mya arenaria*), and the American lobster (*Homarus americanus*). These are the three commer-

cially and recreationally significant species harvested from the bay and are locally resident in the study area for much, if not all, of the year. Clams and lobster from the bay are harvested commercially, and lobster and flounder are harvested recreationally.

Two types of hypothetical seafood consumption scenarios were identified and evaluated in this assessment (Table 2). The first was to represent the maximum-exposed individual (MEI), assumed to consume 165 g/day of seafood. The consumption rate is a default value from the EPA guidance document (2) based on survey data showing that approximately 0.1 % of the U.S. population consumes 165 g/day of seafood. This default value was used in the absence of a definitive consumption survey for the study area. Local interviews confirmed that a small population of recreational fishermen and/or those who rely heavily on self-caught seafood for subsistence are likely to consume this much seafood. Two MEI consumption profiles were evaluated. The mixed diet reflects an individual who catches a large amount of seafood (including clams not caught legally), for home consumption; the flounder diet reflects either local or out-of-state fishermen who keep large enough quantities of Quincy Bay flounder for year-round home consumption.

The second types of consumption scenarios were for typical local consumers (TLC) who were assumed to consume 3.1 g of Quincy Bay seafood/day. The consumption rate was based on New England regional survey data for seafood consumption by species (3). These consumers were considered likely to have regular access to flounder and lobster but not to locally harvested clams. Two profiles for this type of consumer were evaluated, one for the person who eats the lobster tomalley and one for the

levels and hazard identification.^a

Softshell clams			Lobster, tissue			Lobster, hepatopancreas			Flounder, tissue		
Max	Mean	LCD	Max	Mean	LCD	Max	Mean	LCD	Max	Mean	LCD
2.50E-02	2.10E-02	1.70E-02	5.00E-03	2.00E-03	1.00E-03	2.23E+00	1.31E+00	6.93E-01	9.00E-03	1.00E-03	1.00E-03
2.45E-01	2.06E-01	1.67E-01	2.60E-01	2.40E-02	2.00E-03	2.38E+00	7.20E-01	1.03E-01	3.77E-01	2.90E-02	0.00E+00
1.95E+00	1.85E+00	1.76E+00	6.22E+00	4.06E+00	2.67E+00	2.79E+00	1.37E+02	1.77E+01	2.15E-01	1.09E-01	3.60E-02
2.00E-03	2.00E-03	—	1.68E-01	8.50E-02	2.20E-02	1.12E-01	6.50E-02	2.00E-03	8.60E-02	3.00E-02	6.00E-03
4.60E-01	4.50E-01	4.40E-01	2.07E-01	1.69E-01	1.21E-01	7.00E-01	3.35E-01	1.20E-01	4.30E-02	1.50E-02	0.00E+00
3.48E-03	2.88E-03	2.28E-03	6.07E-04	3.76E-04	1.72E-04	2.40E-01	9.75E-02	2.80E-02	3.00E-02	3.15E-03	1.63E-04
1.56E-03	1.26E-03	9.61E-04	1.92E-04	1.66E-04	1.09E-04	8.76E-02	3.18E-02	1.30E-02	7.67E-03	9.14E-04	1.09E-04
1.92E-03	1.62E-03	1.32E-03	4.15E-04	2.10E-04	6.26E-05	1.52E-01	6.57E-02	1.50E-02	2.23E-02	2.24E-03	5.40E-05
1.42E-03	1.23E-03	1.04E-03	6.64E-04	5.28E-04	5.78E-05	3.12E-01	1.00E-01	1.82E-02	1.33E-02	1.58E-03	1.87E-04
4.76E-03	4.26E-03	3.77E-03	7.46E-03	5.03E-03	2.83E-03	1.89E+00	1.30E+00	6.58E-01	1.59E-02	5.19E-03	1.51E-03
3.37E-04	3.03E-04	2.70E-04	6.12E-04	5.45E-04	3.99E-04	7.34E-02	2.95E-02	4.34E-03	4.97E-03	8.55E-04	4.16E-04
1.03E-04	1.02E-04	1.01E-04	2.19E-04	1.33E-04	8.41E-05	1.92E-02	1.37E-02	8.64E-03	2.52E-04	1.27E-04	4.90E-05
1.28E-04	1.19E-04	1.10E-04	1.83E-04	1.63E-04	1.19E-04	3.37E-02	1.84E-02	4.09E-03	8.93E-04	1.82E-04	6.70E-05
1.18E-04	1.17E-04	1.16E-04	1.81E-04	1.22E-04	2.68E-05	3.18E-03	1.78E-03	8.30E-04	1.70E-04	1.56E-04	1.41E-04
4.51E-02	4.35E-02	4.19E-02	7.43E-02	5.19E-02	3.58E-02	4.78E+00	3.37E+00	2.26E+00	2.61E-04	2.45E-04	2.26E-04
1.53E-01	1.51E-01	1.49E-01	3.82E-01	2.37E-01	1.43E-01	6.18E+01	4.39E+01	2.28E+01	7.43E-01	2.73E-01	6.12E-02
1.99E-02	1.44E-02	8.94E-03	3.68E-03	1.97E-03	1.69E-03	2.27E+00	1.50E+00	6.53E-01	1.77E-02	3.61E-03	1.39E+03
1.40E-01	1.36E-01	1.33E-01	3.80E-01	2.37E-01	1.43E-01	5.96E+01	4.24E+01	2.22E+01	7.26E-01	2.71E-01	6.12E-02

observations. IRIS, Integrated Risk Information System; HEA, Health Effects Assessment; Y, data available; N, data unavailable; LCD, lowest concentration

classifications are made without regard to the route of exposure. Route-specific information is considered when determining the carcinogenic potency factor. Public Health Evaluation Manual (13) and US EPA Integrated Risk Information System (14).

Table 2. Summary of assumed lifetime consumption levels.^a

	Maximally exposed individual		Typically exposed individual	
	Mixed diet	Flounder only	Mixed diet	Mixed diet
Quincy Bay clams	16 g/day (26 meals/year)	—	—	—
Quincy Bay flounder	113 g/day (about 182 meals/year)	165 g/day (about 265 meals/year)	1 g/day (1–2 meals/year)	1 g/day (1–2 meals/year)
Quincy Bay lobster ^b				
Tissue	30 g/day (about 115 meals/year)	—	2.1 g/day (6–7 meals/year)	1.7 g/day (6–7 meals/year)
Tomalley	6 g/day (about 115 meals/year)	—	—	0.4 g/day (6–7 meals/year)

^aAssumes 0.5 lb (227 g) serving per meal of clams or flounder and 0.25 lb (113.5 g) serving of edible parts per meal of lobster.

^bBreakdown of tomalley versus other edible lobster tissue based on Massachusetts Division of Marine Fisheries (unpublished data).

person who does not.

The dose calculations were made using the standard assumptions for an integrated EPA risk analysis, including exposure over an entire 70-year lifetime and a 70-kg body weight for an average American adult. In addition, it was assumed in accordance with the EPA Guidance (2) that the ingested dose is equal to the absorbed contaminant dose and that cooking has no effect on the contaminants.

Risk Characterization

To calculate the plausible upperbound to excess lifetime risk of cancer by the EPA methodology, the contaminant-specific dose is multiplied by the EPA CPF for oral exposures to the contaminant. This equation assumes that the slope of the dose-response curve is linear and equal to the CPF. The resulting chemical-specific and species-specific calculated risks are

summed to calculate total upperbound excess lifetime cancer risks.

Results for the estimated maximum upperbound cancer risks from this study of consumption of Quincy Bay seafood are summarized in Tables 3 and 4. Principal conclusions were a) The great majority of the estimated increased cancer risk was attributed to polychlorinated biphenyls (PCBs) in Quincy Bay lobster tomalley for any long-term consumers of even small amounts of this item. b) The only other cancer risks estimated to be greater than 1 in 1000 in this study were associated with the assumed long-term maximum consumption of very large quantities of Quincy Bay flounder, on the order of more than 100 g/day (i.e., about 100 pounds per year) for a 70-year lifetime. c) With the exception of risks due to consumption of lobster tomalley, the estimated risks to the prototype typical local consumers of Quincy Bay seafood were relatively small.

Table 3. Maximum upperbound estimated lifetime cancer risks from consumption of Quincy Bay seafood.^a

	Maximally exposed individual		Typical exposed individual	
	Mixed diet	Flounder only	Mixed diet	Mixed diet
Clams	2.1×10^{-4} ($<1\%$)	—	—	—
Flounder	3.2×10^{-3} (13.9%)	4.7×10^{-3} (100%)	2.8×10^{-5} (33%)	2.8×10^{-5} (2.2%)
Lobster meat	8.0×10^{-4} (3.5%)	—	5.6×10^{-5} (67%)	4.5×10^{-5} (3.5%)
Tomalley	1.9×10^{-2} (82.6%)	—	—	1.2×10^{-3} (92.3%)
Total risk	2.3×10^{-2}	4.7×10^{-3}	8.4×10^{-5}	1.3×10^{-3}

^aPercentage may not equal 100% because of rounding and the need to display no more than two significant digits. Cancer risks calculated using mean contaminant concentrations were 36 to 71% of the maximum values depending on the consumption profile evaluated.

Table 4. Percent contribution to upperbound cancer risk by each organic chemical.^a

Organic compounds ^b	Maximally exposed individual		Typical local consumer	
	Mixed diet	Flounder only	Mixed diet no tomalley	Mixed diet with tomalley
Polyaromatic hydrocarbons (total)	22.84	0.15	30.61	25.55
Polychlorinated biphenyls (total)	76.24	96.99	68.22	73.80
Other compounds ^c	0.92	3.00	1.16	0.65
	100.00	100.00	100.00	100.00

^aFor maximum concentrations only, mean concentrations produced similar results.

^bMetals not included since those included in this study were not considered by EPA to be carcinogenic by ingestion.

^cOther compounds include chlordane, *pp*-DDD, *pp*-DDE, *pp*-DDT, hexachlorobenzene, and hexachlorocyclohexane (alpha and gamma).

Discussion

Comparison with Other Locations and Other Eating and Drinking Activities

The calculated risks of the MEI cases of Quincy Bay seafood consumption in this study were comparable to those calculated in at least two other areas (New York metropolitan area and Lake Michigan) where fishing closures and/or consumption advisories were issued based on PCB levels in seafood (4,5). Adjusting the CPF for PCBs to be the same for the three studies would bring the Quincy Bay, New York area, and Lake Michigan estimates of upperbound increased cancer risk well within a factor of 2 of each other for comparable levels of consumption.

Figure 1 shows results from these studies based on the different CPFs used (2.6 for Quincy Bay and 4.34 for the other two studies). The figure also shows a comparison of the calculated seafood consumption risks with risks calculated by others based on Crouch and Wilson (6) for other types of eating and drinking activities (7). It is recognized that such a comparison tends to obscure potentially legitimate but not fully understood differences in the actual carcinogenic potency of known genotoxic initiators such as aflatoxin B and potential promoters such as PCBs. However, the comparison does illustrate that the calculated MEI seafood consumption risks in this and other studies are one to two orders of magnitude higher than those of the other types of eating and drinking activities commonly discussed in terms of carcinogenic risk. Even for the hypothetical typical local consumer of Quincy Bay seafood, the calculated upperbound cancer risks of consuming a mixed diet of locally caught seafood inclusive of six to seven meals per year of lobster tomalley are up to ten times higher than the calculated cancer

risks of the other eating and drinking activities. However, without lobster tomalley, the Quincy Bay seafood consumption risks for the typical local consumer drop into the 10^{-4} to 10^{-5} range characteristics of those calculated for many of the other eating and drinking activities.

Uncertainties

Extreme caution should be exercised in the interpretation and use of any seafood consumption risk assessment results due to a variety of uncertainties. Specific sources of uncertainty illustrated by the Quincy Bay risk assessment are discussed below.

Representativeness of the Measured Values for Contaminants. Comparison of the 1987 EPA Quincy Bay data for contaminant residues in seafood (1) with other recent data for the same species from Massachusetts waters suggests that the EPA data are representative for Quincy Bay, given the differences in sample locations and analytical methods among studies. In particular, an interlaboratory comparison of PCB levels in edible lobster tissues among EPA, MA Division of Marine Fisheries and U.S. FDA scientists (8) showed similar and internally consistent results.

However, if one goes beyond the tissue residue data to examine potential correlations to sediment contamination (and remedial action considerations), the question arises of the representativeness of the typically available sediment contaminant data. As shown in Figure 2, PCB levels in both sediments and seafood from Quincy Bay are consistently two to six times higher than those from certain offshore areas of Massachusetts Bay. There is reason to believe both that sediments are a major source of exposure to the contaminants (9), but that the sediments sampled are not the only exposure

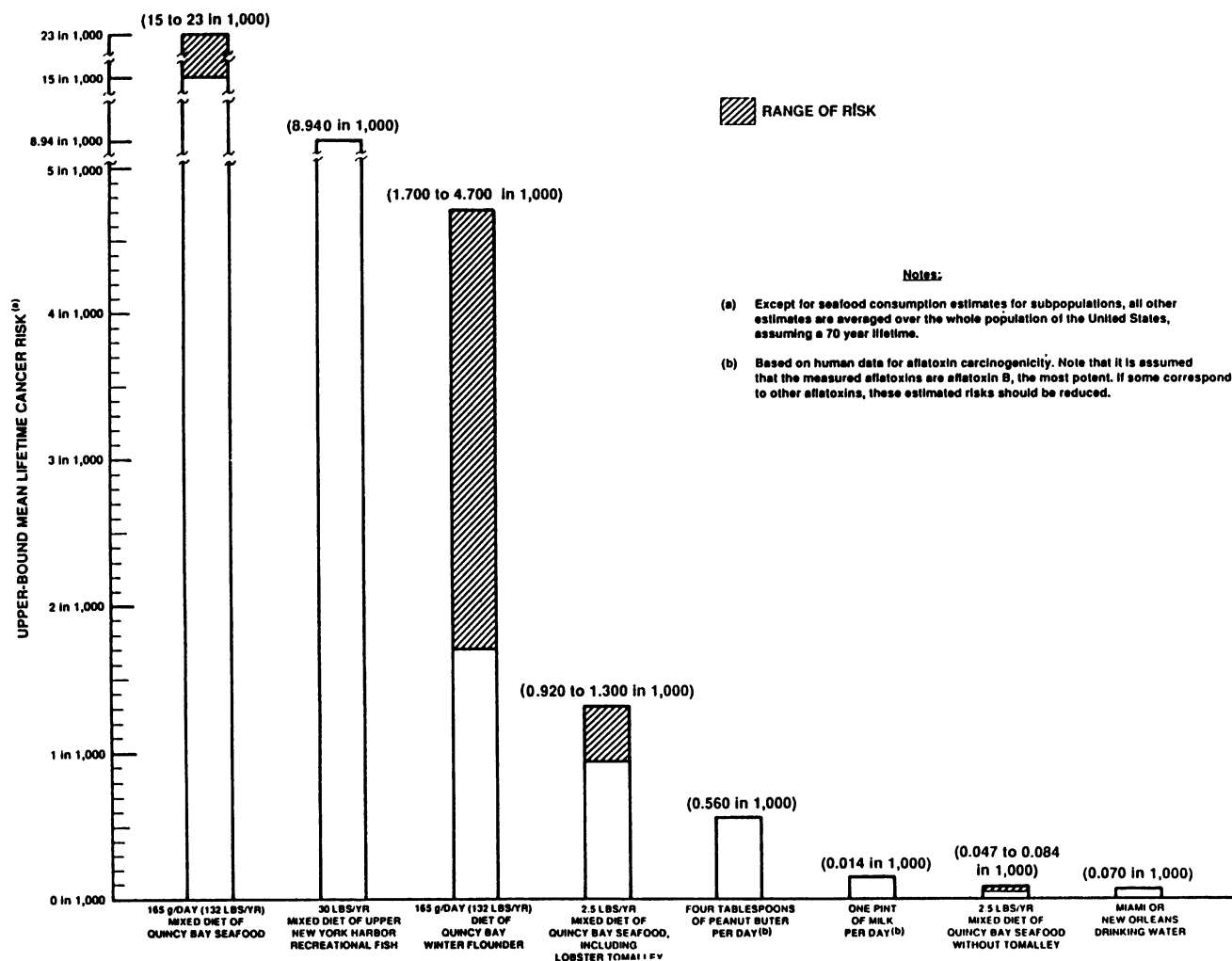


FIGURE 1. Comparison of estimated lifetime cancer risks (plausible upper limit) associated with various eating and drinking activities. Modified from Meta Systems, Inc. (1), Clark et al. (4), and Belton (5).

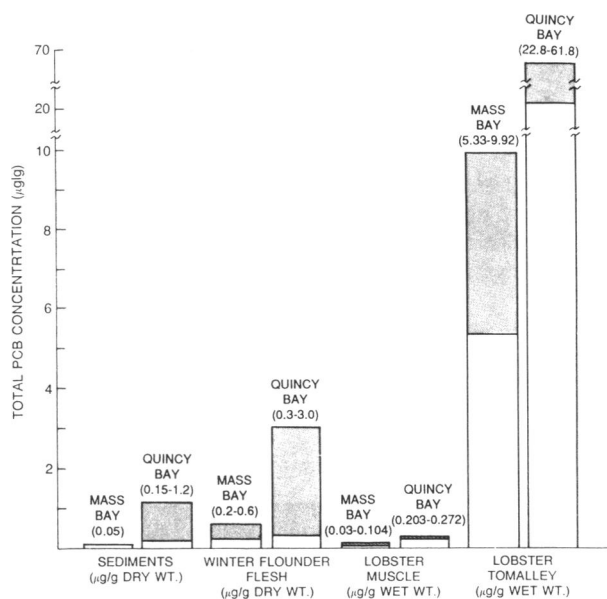


FIGURE 2. Comparison of PCBs in organisms and sediments from two locations in 1987. From MWRA (11), Pruett et al. (8), and Gardner and Pruett (1).

sources of interest. In addition to the sediments sampled in this study, there are those permanently suspended or more frequently resuspended sediments known to occupy the lower water column in locations like the study area, but these are rarely sampled by conventional techniques. What are the levels of contamination in these suspended sediments, and how much do they redistribute in time and space relative to patterns of seafood movement? If one isolates seafood from the deposited sediments (e.g., by capping or removing the sediments), is the remaining exposure to contaminants in suspended sediment still enough to allow residues of concern to build up in the seafood?

Use of Standard EPA Risk Assessment Assumptions. Many of the assumptions contained in the EPA guidance (2) and used in the EPA Quincy Bay study are standard EPA risk assessment assumptions chosen to be conservative in view of uncertainty. These include the following assumptions: a) use of a linearized, multistage model of carcinogenesis with the best available CPF. This approach does not distinguish fully among chemicals whose genotoxicity or whose roles as initiators or promoters in humans are not fully understood. On the one hand, one would expect that

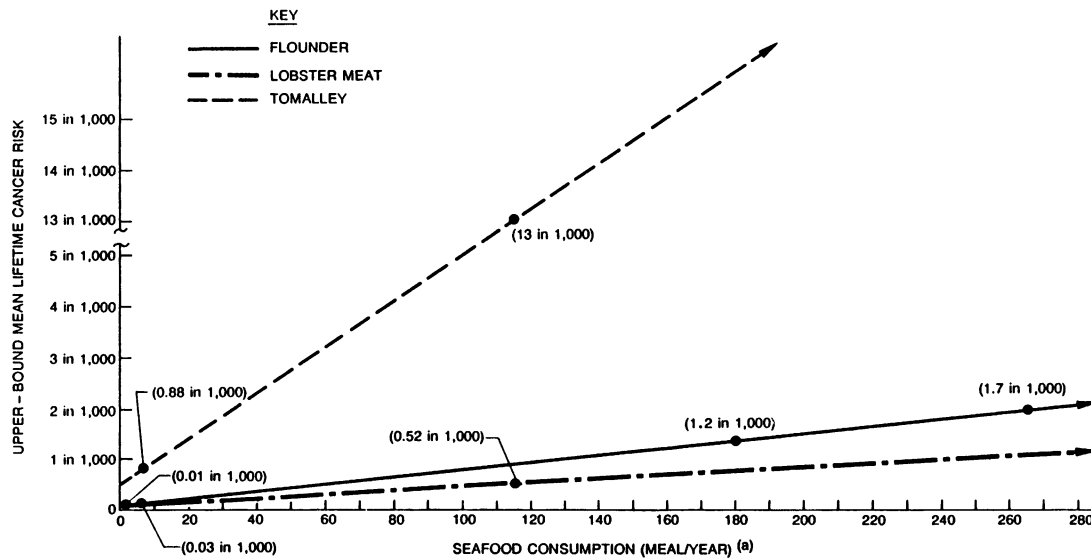


FIGURE 3. Estimated cancer risk at different levels of consumption of Quincy Bay seafood. See Table 2 for assumed size of meals.

this assumption is very conservative because it ascribes extreme carcinogenic potential to compounds and congeners for which such toxicity has not been demonstrated. On the other hand, in this case there are some other factors that may suggest that such conservatism is appropriate. First, some chemicals suspected of playing a role in human carcinogenesis were likely present but not analyzed for in the Quincy Bay seafood. For example, 2,3,7,8-TCDD was not analyzed for in this study but was detected in lobsters from offshore dump sites in the New York Bight at levels high enough to produce estimates of about 1.5×10^{-3} plausible upperbound increased cancer risk from consumption of 50 lobsters per year (10). Second, at least one element of the seafood in question here delivers a mix of suspected promoters and initiators of carcinogenesis together in a lipid-rich medium (lobster tomalley), which may have a net carcinogenic potential quite different from that of the experimental systems from which the results were obtained to generate the standard CPFs used here. For example, all five of the polynuclear aromatic hydrocarbons (PAHs) rated by EPA as having sufficient evidence of animal carcinogenicity were detected, along with high PCB levels in lobster tomalley in this study (Table 1).

b) Assumption that cooking and/or other factors do not change available contaminant levels to the consumer. This also includes the assumption that the ingested contaminant dose is totally absorbed. Again, one can presume that these are generally conservative and potentially unrealistic assumptions. However, there is also the question in this case of whether the boiling of lobsters in traditional fashion (whole, without disturbance of the body cavity) results in migration of contaminant from the tomalley to other edible portions (tail and/or body meat) eaten by the great majority of lobster consumers.

Affected Population Size and Consumption Patterns. Figure 3 illustrates a sensitivity analysis of the calculated upperbound increased cancer risks from the Quincy Bay study as a function

of amount of seafood consumed by species. Estimates of the actual size of affected consumer populations were not made in the EPA study due to the necessary reliance on a fall-winter study period when original recreational fisheries survey data could not be collected. Based on interviews, there is a likelihood that some Quincy area residents consume amounts of locally caught seafood in between the amounts used as maximum and typical in the calculations. In addition, many actual consumers of Quincy Bay seafood also consume varying amounts of seafood taken from other locations with contaminated sediments but for which seafood residue data are not yet available. For example, PCB levels in sediments from the EPA Quincy Bay study may be compared with similar and in some cases higher levels measured near other major wastewater outfalls in Boston Harbor (11).

Transfer Pathways of Pathogens in Seafood. The task II and task III work in the Quincy Bay study (1) identified pathological symptoms in flounder and clams of uncertain significance to human consumers. Particular uncertainty exists as to the extent, if any, to which viral pathogens may remain in and affect human consumers of clams, even following required bacteriological depuration. This subject area is one where much fundamental research has yet to be implemented.

Implications

Taken together, the results and uncertainties of the EPA Quincy Bay study have a number of implications. To reduce uncertainty about the validity of basic seafood risk assessment assumptions such as those in the EPA guidance (2) and to investigate pathogen transfer, some additional laboratory studies would be helpful. Faced with somewhat similar uncertainties in evaluating the net toxicity to aquatic life of complex effluents, the concept of whole effluent toxicity testing was developed and is being widely implemented. Perhaps a concept of representative seafood consumption bioassay is needed. For example, feeding

studies that concentrate on delivery of contaminated seafood in representative form (cooked or raw) in appropriately controlled experimental systems could help provide better understanding of many of the uncertainties discussed here. Realistic compromises would need to be reached concerning doses, mixtures, exposure timeframes, etc., so that such studies would be deemed practical and useful in the context of other accepted techniques in cancer research and pathogen transmittal.

Given the evidence that additional areas of seafood harvest exhibit sediment contaminant levels similar to those found in Quincy Bay by the EPA study, a two-pronged approach to additional monitoring of contaminant residues in seafood may be appropriate. One component of such an approach would require the analysis of contaminant residues in those major seafood species from the contaminated sites that spend enough time at those sites to be at risk of contamination. The second component would involve greatly expanded monitoring of contaminant residues in dockside landings destined for local markets. Taken together, the data from these types of monitoring would provide a much better basis for exposure considerations in refined risk assessments and for appropriately targeting public health advisories, either recreational or commercial harvesting restrictions, and remedial action priorities for contaminated areas.

The feasibility of risk reduction through remedial action can be tested in locations such as Quincy Bay where risk concerns have been documented and *in situ* experimental remediation is feasible. In particular, as soon as the major wastewater discharges to the bay are ended in the 1990s, a focused investigation of the reduction (if any) of localized seafood contaminant levels in conjunction with sediment manipulation experiments could take place. Such a study would help demonstrate whether such manipulation techniques as removal or reverse layering of deposited sediments result in measurable improvements in seafood residue levels, or whether less controllable suspended sediments continue to pose a longer term source of significant contaminant exposure.

Regulatory Response

Agencies participating in the Quincy Bay study included EPA, U.S. Food and Drug Administration (FDA), and National Marine Fisheries Service (NMFS), Massachusetts agencies, including the Office of Coastal Zone Management (MCZM), Department of Public Health (MDPH), Division of Marine Fisheries (MDMF), Department of Environmental Quality Engineering (DEQE), and Massachusetts Water Resources Authority (MWRA), and others including the City of Quincy Department of Public Health and outside academic experts.

The Quincy Bay study risk assessment was selected as the first case study of the newly formed EPA/FDA Fish Contamination Committee. This committee was created as a result of discussions held between EPA and FDA to attempt to resolve differences in risk assessment jurisdiction and methodology. Guidance was sought from this committee on several risk assessment issues including jurisdictional issues, the appropriate CPF for the mix of congeners found in Quincy Bay biota, appropriateness of analytical methodology, consumption values, and comparability of Quincy Bay risk numbers to the FDA PCB tolerance. As a result of the request, the EPA Office of Health and Environmental Assessment (OHEA) reviewed the PCB congener data from

Quincy Bay and developed a congener-specific CPF based on the Aroclor congener mix most similar to that found in the Quincy Bay samples.

As a result of the Study findings, EPA developed a series of recommendations, some requiring implementation on both national and local levels. EPA met with Massachusetts agencies to develop consensus and to determine responsibility for recommendations that would be implemented by the appropriate agency. The highest priority recommendations with the responsible agencies in parentheses are summarized as follows: issue health advisories against consumption of lobster tomalley and seafood from urban areas (MDPH); develop an educational program to communicate the risk from consumption of contaminated seafood (MDPH, EPA); expand regulatory oversight and monitoring of seafood (FDA, MDPH); establish restrictions on seafood harvest near wastewater discharges in urban areas (MDPH, MDMF); develop formal risk assessment methodology, regulatory guidance limits for priority seafood contaminants, and standard methodologies for measurement of chemical contaminants in seafood (FDA, EPA); and develop ambient sediment quality criteria (EPA). Other recommendations included further research on cooking effects, the relationship between organism pathology, chemical contaminants, and human health risk, CPF development, and monitoring programs in Quincy Bay and in the rest of Boston Harbor.

Most recommendations of the study have been or are in some state of implementation. For instance, concurrent with release of the study, MDPH issued the recommended advisories and since has been attempting to expand its seafood monitoring program. MDPH has also been working with EPA to develop both short-term and long-term education programs.

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